# Scientists discover key to what causes immune cell migration to wounds

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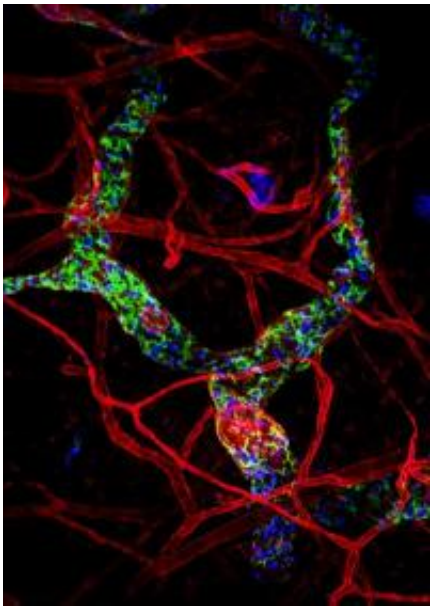

This is a microscopic image of blood vessels (red), lymphatic vessels (green) and Chemokin CCL21 (blue), marked with different fluorescent colors. © IST Austria/Sixt group

Immune cells play an important role in the upkeep and repair of our bodies, helping us to defend against infection and disease. Until now, how these cells detect a wounded or damaged site has largely remained a mystery. New research, led by University of Bristol academics in collaboration with a team from the University of Sheffield, has identified the triggers which lead these cells to react and respond in cell repair.

It is hoped the findings, published in *Current Biology*, could help scientists design therapies to manipulate the cell repair process and direct immune cells away from sites where they are doing damage, such as tumours, and send them to places where they are needed.

Previous studies had found that the earliest signal produced at a wound site responsible for attracting immune cells to the damaged site is hydrogen peroxide ($H_2O_2$). However, it was still unclear how these cells detect this chemical, and what signaling occurs in these cells downstream of $H_2O_2$ detection to power their rapid migration.

Using the common fruitfly (*Drosophila melanogaster*) and timelapse microscopy, the team led by Professor Will Wood at the University of Bristol were able to study the process in situ and identify what causes the cells to migrate to sites of damage where they then detect, ingest and degrade debris, dying cells and invading pathogens.

After dissecting the signaling occurring in immune cells responding to wound induced ($H_2O_2$), the team found that it involved a well-established immune signalling pathway used in vertebrate adaptive immune responses. The results suggest that adaptive immune signalling pathways important in distinguishing self from non-self in vertebrates appear to have evolved from a more ancient response designed to distinguished 'damaged self' from 'healthy self'.

Will Wood, Professor of Developmental Biology, in Bristol's School for Cellular and Molecular Medicine, and the study's lead author, said: "While inflammation is critical to prevent infection, too much of a response by immune cells can cause or worsen a wide range of human diseases and conditions including autoimmunity, atherosclerosis, cancer and chronic inflammation.

"This research is therefore critical for improving human health as it

enables us to discover novel points of intervention to manipulate immune cell behaviour and allow us to design therapies to direct immune cells away from sites where they are doing damage and send them into places where they are needed."

**More information:** Draper/CED-1 mediates an ancient damage response to control inflammatory blood cell migration in vivo by Iwan Robert Evans, Frederico Salgueiro Lopes Matias Rodrigues, Emma Louise Armitage and Will Wood in *Current Biology*.

Provided by University of Bristol